CASE REPORTS

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Meningoencephalitis Due to Infectious Mononucleosis

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IN THE PAST FEW YEARS the occurrence of central nervous system involvement in infectious mononucleosis has been noted in the literature with increased frequency.[1, 2, 4, 5] The estimated incidence of this complication is probably less than one per cent, but the mortality rate due to central nervous system involvement is apparently high. Lawrence[3] noted that neurological complications were the cause of death in seven of sixteen reported fatal cases of infectious mononucleosis. Any part of the central nervous system may be affected. The report herewith brings to five the number of reported cases of acute meningoencephalitis complicating infectious mononucleosis.

REPORT OF A CASE

The patient, a 24-year-old white man, was apparently well until May 7, 1954, when mild retro-orbital headache, malaise, anorexia, and a feeling of lightheadedness developed. The symptoms persisted but the patient was able to continue attending college classes. On May 12, 1954, he noted the onset of chilliness and excessive perspiration and on May 13 he was admitted to the hospital.

The general health of the patient had been excellent and, except for an injury of the neck incurred in a fall at the age of 18, he had had no serious illness.

At the time of admittance the temperature was 98.8° F., the pulse rate 90, respirations 20 per minute and the blood pressure 112/84 mm. of mercury. There were scattered acniform areas over the upper back. The vessels in the pharynx were slightly engorged and there were two small spots of white exudate on the right tonsil. Scattered lymph nodes about 0.5 cm. in diameter were palpated in both the cervical and the axillary areas. A prominent epitrochlear node was present on the right. The remainder of the examination was within normal limits.

On the first hospital day the temperature was 99.4° F. A feeling of light-headedness still was present and the patient vomited twice after the evening meal. Results of a neurological examination at this time were again normal.

On the second hospital day at 6:20 a.m. the patient suddenly had generalized clonic and tonic convulsions without incontinence, lasting some thirty seconds, following which he was semicomatose. Thirty minutes later generalized convulsion occurred again, characterized by violent athetoid motions of all extremities, wandering divergent eye movements and incontinence. This episode lasted some three hours. Sedation was parenterally administered and the patient gradually subsided into a comatose, restless state. There were no signs of meningeal irritation or paralysis. The reflexes were hyperactive but not of pathological order. The temperature rose to 104° F. (rectal). The patient remained comatose for approximately 48 hours. Moderate nuchal rigidity developed within ten hours after the convulsion and abdominal and cremasteric reflexes were absent; as extensor plantar responses were present on both sides.

On the fourth hospital day the temperature was 99.6° F. and the patient began to respond drowsily to simple questions. In the succeeding 24 hours he gradually became more alert, although still lethargic and slurring and slow of speech. From this point on, improvement was rapid. The abdominal and cremasteric reflexes returned and extensor plantar response had disappeared by the sixth hospital day. The generalized lymphadenopathic condition was more pronounced but the minimal spotty exudate in the pharynx had subsided. Nuchal stiffness persisted to some degree until the twelfth hospital day. At this time for a four-day period the patient noted polydipsia and polyuria, with an oral intake of 7,200 cc. of fluids on one day and output of 5,000 cc. of urine. The specific gravity of the urine during this transient phase was 1,001. These symptoms subsided spontaneously and the patient was discharged on the nineteenth hospital day without residual effect of the severe illness except for a generalized lymphadenopathic condition.

Results of blood, urine and spinal fluid studies are listed in Table 1. No bacterial growth was obtained on either blood or spinal fluid cultures. Results of skin tests with first strength purified protein derivative and 1:100 dilutions of coccidioidin were

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TABLE 1.—*Laboratory data in case of meningoencephalitis*

	May 13	May 15	May 16	May 18	May 20	May 24	May 28
Blood							
Hemoglobin (gm. per 100 cc.)	13.8						13.6
Leukocytes (per cu. mm.)	6,900	15,400	11,000	11,000	12,200	11,000	7,200
Neutrophils per cent:							
Segmented	31	69	46	30	29	34	66
Non-segmented	7	10	16	6	9	14	8
Lymphocytes	62	12	35	64	60	45	25
Atypical (per cent)	70	50	90	70	50	80	30
Monocytes			3				
Eosinophils					2		
Heterophil titer (guinea pig absorption)	1:10			1:20	1:80	1:80	1:160
Sedimentation rate (mm. per hour Wintrobe)	7					16	7
Cerebrospinal fluid							
Appearance		clear		clear			
Pressure (mm. water)		110		135			
Cells							
Leukocytes (cu. mm.)		10		6			
Lymphocytic (per cent)		80		5			
Sugar (mg. per cent)		108		73			
Protein (mg. per cent)		175		90			
Gold curve		1112231100		0001110000			
Wassermann				Neg.			
Chlorides (mg. per cent)				638			
Heterophil antibody titer				1:10			

negative. Complement fixation studies on the serum for western equine encephalitis, St. Louis encephalitis and mumps were negative. Electrolyte studies of the serum during the phase of polyuria were within normal limits. No abnormalities were noted in liver function studies. Hemolytic staphylococcus aureus grew on cultures of material taken from the throat.

COMMENT

The cause of infectious mononucleosis is unknown, but the concept of a virus as the infecting agent is generally held by most authorities. The pronounced variability from case to case in symptomatology, physical findings and the duration of illness make this a most bizarre disease. If central nervous system symptoms are present, they may appear at the onset, although most commonly they do not occur until one to three weeks after onset.

In the case reported upon herein, the central nervous system manifestations appeared during the first week of his illness. The appearance of the pharyngeal exudate and the enlargement of lymph nodes were initially suggestive of the diagnosis. The confirmatory laboratory studies and the rising heterophil titer substantiated it. The spleen was never palpable.

The clinical picture produced by involvement of the nervous system may be indistinguishable from that caused by many other factors. Since the systemic signs of infectious mononucleosis may be minimal, it is important to realize the value of heterophil antibody tests in obscure cases of central nervous system symptomatology.

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Limited Chronic Tension Pneumothorax with Lobar Atelectasis

Two Cases Treated by Lobectomy and Decortication

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CHRONIC PNEUMOTHORAX with positive pressure secondary to a pulmonopleural or bronchopleural fistula of valvular type occurs in many conditions[10] such as tuberculosis, pulmonary suppuration of other types, spontaneous rupture of emphysematous blebs and in empyemas in which the pus is coughed up and does not reform because of antibiotic therapy. Usually the involved lung is more or less uniformly collapsed but occasionally previous pleural symphysis limits the extent of pneumothorax so that collapse is localized to one or more lobes of a lung.

Two patients affected by limited tension pneumothorax with complete lobar collapse and suppuration were observed by the author within a month and both were treated by lobectomy and decortication.